

Editorial

OMICS Strategy Discoveries to Obesity in the Prevention and Personalized Therapy

Kim HD*, Carpenter ML and Heck DE

Department of Environmental Health Sciences, New York Medical College, USA

*Corresponding author: Hong Duck Kim, Department of Environmental Health Sciences, School of Health Science and Practice, New York Medical College, Valhalla, NY 10595, USA

 $\textbf{Received:} \ \text{May 02, 2016;} \ \textbf{Accepted:} \ \text{April 06, 2016;}$

Published: May 10, 2016

Editorial

Obesity (BMI > or = 30) rated as the second leading cause of death and an increasing socioeconomic burden such a public related aspects with a mortality rate that is likely 2.6 million people each year according to WHO. Obesity contributes to metabolic disorders causing imbalanced energy combined with genetic defects including the brain reward circuit and metabolic impairment [1-4]. The prevalence of obesity may associate with environmental chemical exposure, which affects fat mass and adipogenesis in the United States [5]. However, it is poorly understood that the effects of several factors, for example, a wide range of stresses and medical over usage such as birth control pills, antidepressants, and antipsychotics reflects tissue damage and intervenes with the neuronal circuit due to evoked oxidative stress resulting from the interaction between macromolecules such as DNA, lipid and protein. Also including a wide range of environmental factors such as chemical ingredients found in a variety of foods and overburden stresses like the absence of smoking, pregnancy, menopause, sleeping disorder, anxiety, and respiratory problems which occur in everyday life. In addition, some reports indicate that obesity is associated with brain disorder like as malfunction of brain reward system against stimulatory impulse or responses. The mechanism underlying the molecular "trigger" for prevention and therapy in prominent metabolic disorder such as Obese and diabetes still remains to be a mystery. Interestingly, there are many evidences to support interaction between obesity and several types of disorders for example, dysglycemia, dyslipidemia, CVD, stroke, hypertension, and cancer including endometrial, breast, prostate, colon, and GI cancer [6-12]. Advent of Cutting edge knowledge derived from system biology and integration of genomics study such as GWAS and NGS- based detection tools can apply to diagnose and predict diseases status using clinic samples which may contain multilayer formulated macromolecules from patients. Both analytical tools could be optimized for precision of medicine for obesity and extended health complications and causes such as Cardiovascular Disorder (CVD) for atherogenic dyslipidemia and diabetes for insulin resistance on abdominal obesity as multiple metabolic risk factors. Prior to using genomics based analytical tools, analytic platform consist of several modules based on target molecules with various disciples of the study of Omics categories including

Proteomics, Transcriptomics, Metabolomics, Cellomics, Lipidomics, Glycomics, Phamcogenomics, Neurogenetics and Nutrigenomics [13,14]. The output give us a better understanding of what metabolic syndromes are related to the development of disease pathogenesis in molecular levels such as obesity in which risk factors in network/cluster of molecules may interrupt by triggers (stressors) result from internal and external factors in various sources like environmental, genetic, and metabolic. Among them, neurogenetics and nutrigenomics are considered to describe which part of brain would modify stimulus to intake bad foods (poor nutrition) as energy resources or expenditure to resist or survival along with molecular networks in several organ interactions under brain circuits. Those omics approach may lead to provide solutions which brain circuit or interaction molecule network in the food and receptors on the tissue or organ while stress stimulate take food more as compensatory mechanism. To visualize an improved detection system in the tissue and monitoring brain reward cascade between brain and adipose tissue, better and earlier diagnostic tools such as Omics oriented strategy make a chance for success in treatment or provide personalized therapy for individuals.

Nutrigenomics is a study of the role of nutrients on how gene expression could be benefit to understand the cause of obesity and to determine underlying factors associated with various metabolic diseases due to molecular dysfunction in the mice and human body [15,16]. By using new technologies in the field of Omics, we can better visualize the molecular roadmap in metabolic diseases and gain better solutions by establishing a higher degree of dimensionality to understand the various deleterious effects of outside influences (treatment). Moreover, it gives us a better direction in risk management such as how food contamination from bacterial infection or food ingredients, chemical preservatives becomes oxidative stressors to defer impairment of homeostatic networks including the interruption of brain reward with specific dopaminergic receptor and abnormal metabolic cascade causing insulin resistance [17].

Oxidative stress occurs when cellular repair systems cannot readily detoxify ROS. Although ROS generation occurs during normal cellular metabolism within the mitochondria to generate energy, oxidative stress occurs when there is an imbalance of redox system which may lead to ROS-mediated toxicity linked damage with a variety of biological macromolecules includes lipid, DNA, and protein. Concordantly, ROS are key initiators of cellular signaling cascades that lead to various metabolic disorders such as Insulin Resistance (IR) and fasting hyperinsulinemia, which may be associated with mitochondrial dysfunction and oxidative stress [18]. Similar studies have indicated that the oxidative stress (i.e., ROS and RNS) produced by exogenous or endogenous factors influences complex dynamics of human behavior associated with health disparities like lack of vitamin D and exercise which should be of primary concern pertaining to endothelial dysfunction, systemic

inflammation, and chronic hypertension by epigenetic and post-translational modification [19-24]. Nutrigenomic and epigenetic studies are such a double-edged sword of discovery tool which generally focuses on dietary patterns according to genetic variations, the role of gene-nutrient interactions, gene-diet-phenotype interactions and epigenetic modifications caused by nutrients; these molecular tools will facilitate an understanding of the early molecular events that occur in diabetes and will contribute to the identification of better biomarkers and diagnostics tools. Omics approach could help to develop tailored diets that maximize the use of nutrients and cumulated functional ingredients present in good food, which should be treating aberrant adipose-like stem cell in the prevention Obese and delay of diabetes associated with other health complications.

In the future era, we consider that omics trial equipped with molecule based tools by stratification such as nutrigenomics, metabolomics, and neurogenetic research promises to discover a key knowledge of biological function which molecules may involve the pathogenesis network or compulsory individual response to diet pattern in alteration of brain circuit, which is also important to monitor environmental factors that interacts with molecule regulator such as microRNA in epigenetic area could lead to modulate disease risk [25-29]. A clear understanding of these interactions may drive to support the concept of disease prevention through stratification of risk pattern in personal medicine as well as optimization of dietary recommendations. Current research progress has been started to provide a resolution rapidly in the metabolic disorders including obesity, where specific targeted nutritional advice, such as a Mediterranean diet, helps to decrease cardiovascular risk factors and stroke incidence in people with polymorphisms strongly associated with type 2 diabetes. Omics is impacted to promote greater innovation, which encompass harness of stress from the environment to prevent metabolic disorders as well as improve quality of life with the health of food through different angles of approaches equipped with new detection modules such as protein array, NGS, metagenome, which will provide a framework for the development of genome-dependent food control for health brain reward strategies and the personalized approaches for the prevention and management of diabetes mellitus, stroke and CVD in public health.

References

- Kenny PJ. Reward mechanisms in obesity: new insights and future directions. Neuron. 2011; 69: 664-679.
- Loskutoff DJ, Fujisawa K, Samad F. The fat mouse. A powerful genetic model to study hemostatic gene expression in obesity/NIDDM. Ann N Y Acad Sci. 2000: 902: 272-281.
- Phillips C, Lopez-Miranda J, Perez-Jimenez F, McManus R, Roche HM. Genetic and nutrient determinants of the metabolic syndrome. Curr Opin Cardiol. 2006: 21: 185-193.
- Logan M, Der Merwe MTV, Dodgen TM, Myburgh R, Eloff A, Alessandrini M, et al. Allelic variants of the Melanocortin 4 receptor (MC4R) gene in a South African study group Mol Genet Genomic Med. 2015; 4: 68-76.
- Buckley JP, Herring AH, Wolff MS, Calafat AM, Engel SM. Prenatal exposure to environmental phenols and childhood fat mass in the Mount Sinai Children's Environmental Health Study. Environ Int. 2016; 91: 350-356.
- Lin JE, Colon-Gonzalez F, Blomain E, Kim GW, Aing A, Stoecker B, et al. Obesity-Induced Colorectal Cancer Is Driven by Caloric Silencing of the Guanylin-GUCY2C Paracrine Signaling Axis. Cancer Res. 2016; 76: 339-346.

- Arnold M, Colquhoun A, Cook MB, Ferlay J, Forman D, Soerjomataram I.
 Obesity and the Incidence of Upper Gastrointestinal Cancers: An Ecological Approach to Examine Differences across Age and Sex. Cancer Epidemiol Biomarkers Prev. 2016; 25: 90-97.
- Benn M, Tybjærg-Hansen A. High body mass index and cancer risk-a Mendelian randomisation study. Eur J Epidemiol. 2016.
- Aung K, Lorenzo C, Hinojosa MA, Haffner SM. Risk of Developing Diabetes and Cardiovascular Disease in Metabolically Unhealthy Normal-Weight and Metabolically Healthy Obese Individuals. J Clin Endocrinol Metab. 2014; 99: 462-468.
- Weiss R, Magge SN, Santoro N, Giannini C, Boston R, Holder T, et al. Glucose Effectiveness in Obese Children: Relation to Degree of Obesity and Dysglycemia. Diabetes Care. 2015; 38: 689-695.
- Putnam K, Shoemaker R, Yiannikouris F, Cassis LA. The renin-angiotensin system: a target of and contributor to dyslipidemias, altered glucose homeostasis, and hypertension of the metabolic syndrome. Am J Physiol Heart Circ Physiol. 2012; 302: H1219-H1230.
- Atkins JL, Whincup PH, Morris RW, Lennon LT, Papacosta O, Wannamethee SG. Sarcopenic obesity and risk of cardiovascular disease and mortality: a population-based cohort study of older men. J Am Geriatr Soc. 2014; 62: 253-260.
- 13. Barh D, Blum K, Madigan MA. Omics: Biomedical Perspectives and Applications. Boca Raton, FL; CRC Press. 2010.
- Barcelo D. Wilson & Wilson's Comprehensive Analytical Chemistry: Fundamentals of Advanced Omics Technologies: from Genes to Metabolites: From Genes to Metabolites. Oxford; Elsevier. 2014.
- 15. Waller-Evans H, Hue C, Fearnside J, Rothwell AR, Lockstone HE, Caldérari S, et al. Nutrigenomics of High Fat Diet Induced Obesity in Mice Suggests Relationships between Susceptibility to Fatty Liver Disease and the Proteasome. PLoS One. 2013; 8: e82825.
- 16. Pasman WJ, Van Erk MJ, Klöpping WAA, Pellis L, Wopereis S, Bijlsma S, et al. Nutrigenomics approach elucidates health-promoting effects of high vegetable intake in lean and obese men. Genes Nutr. 2013; 8: 507-521.
- Eisenstein SA, Gredysa DM, Antenor-Dorsey JN, Green L, Arbeláez AM, Koller JM, et al. Insulin, Central Dopamine D2 Receptors, and Monetary Reward Discounting in Obesity. PLoS One. 2015; 10: e0133621.
- Banse HE, Frank N, Kwong GPS, McFarlane D. Relationship of oxidative stress in skeletal muscle with obesity and obesity-associated hyperinsulinemia in horses. Can J Vet Res. 2015; 79: 329-338.
- Zuk A, Fitzpatrick T, Rosella LC. Effect of Vitamin D3 Supplementation on Inflammatory Markers and Glycemic Measures among Overweight or Obese Adults: A Systematic Review of Randomized Controlled Trials. PLoS One. 2016: 11: e0154215.
- Lobato NS, Filgueira FP, Akamine EH, Tostes RC, Carvalho MH, Fortes ZB. Mechanisms of endothelial dysfunction in obesity-associated hypertension. Braz J Med Biol Res. 2012; 45: 392-400.
- Radak Z, Zhao Z, Koltai E, Ohno H, Atalay M. Oxygen Consumption and Usage During Physical Exercise: The Balance Between Oxidative Stress and ROS-Dependent Adaptive Signaling Antioxid Redox Signal. 2013; 18: 1208-1246.
- Savini I, Catani MV, Evangelista D, Gasperi V, Avigliano L. Obesity-Associated Oxidative Stress: Strategies Finalized to Improve Redox State. Int J Mol Sci. 2013; 14: 10497-10538.
- Assies J, Mocking RJT, Lok A, Ruhé HG, Pouwer F, Schene AH. Effects
 of oxidative stress on fatty acid- and one-carbon-metabolism in psychiatric
 and cardiovascular disease comorbidity. Acta Psychiatr Scand. 2014; 130:
 163-180.
- Marseglia L, Manti S, D'Angelo G, Nicotera A, Parisi E, Rosa GD, et al. Oxidative Stress in Obesity: A Critical Component in Human Diseases. Int J Mol Sci. 2015; 16: 378-400.
- Blum K, Thanos PK, Gold MS. Dopamine and glucose, obesity, and reward deficiency syndrome. Front Psychol. 2014; 5: 919.

Hong Duck Kim

Austin Publishing Group

26. Fornaro M, Gabrielli F, Albano C, Fornaro S, Rizzato S, Mattei C, et al. Obsessive-compulsive disorder and related disorders: a comprehensive survey. Ann Gen Psychiatry. 2009; 8: 13.

- 27. Roth CL, Hinney A, Schur EA, Elfers CT, Reinehr T. Association analyses for dopamine receptor gene polymorphisms and weight status in a longitudinal analysis in obese children before and after lifestyle intervention. BMC Pediatr. 2013; 13: 197.
- Bouret S, Levin BE, Susan E. Ozanne Gene-Environment Interactions Controlling Energy and Glucose Homeostasis and the Developmental Origins of Obesity Physiol Rev. 2015; 95: 47-82.
- 29. Mang GM, Pradervand S, Du NH, Arpat AB, Preitner F, Wigger L, et al. A Neuron-Specific Deletion of the MicroRNA-Processing Enzyme DICER Induces Severe but Transient Obesity in Mice. PLoS One. 2015; 10: e0116760.